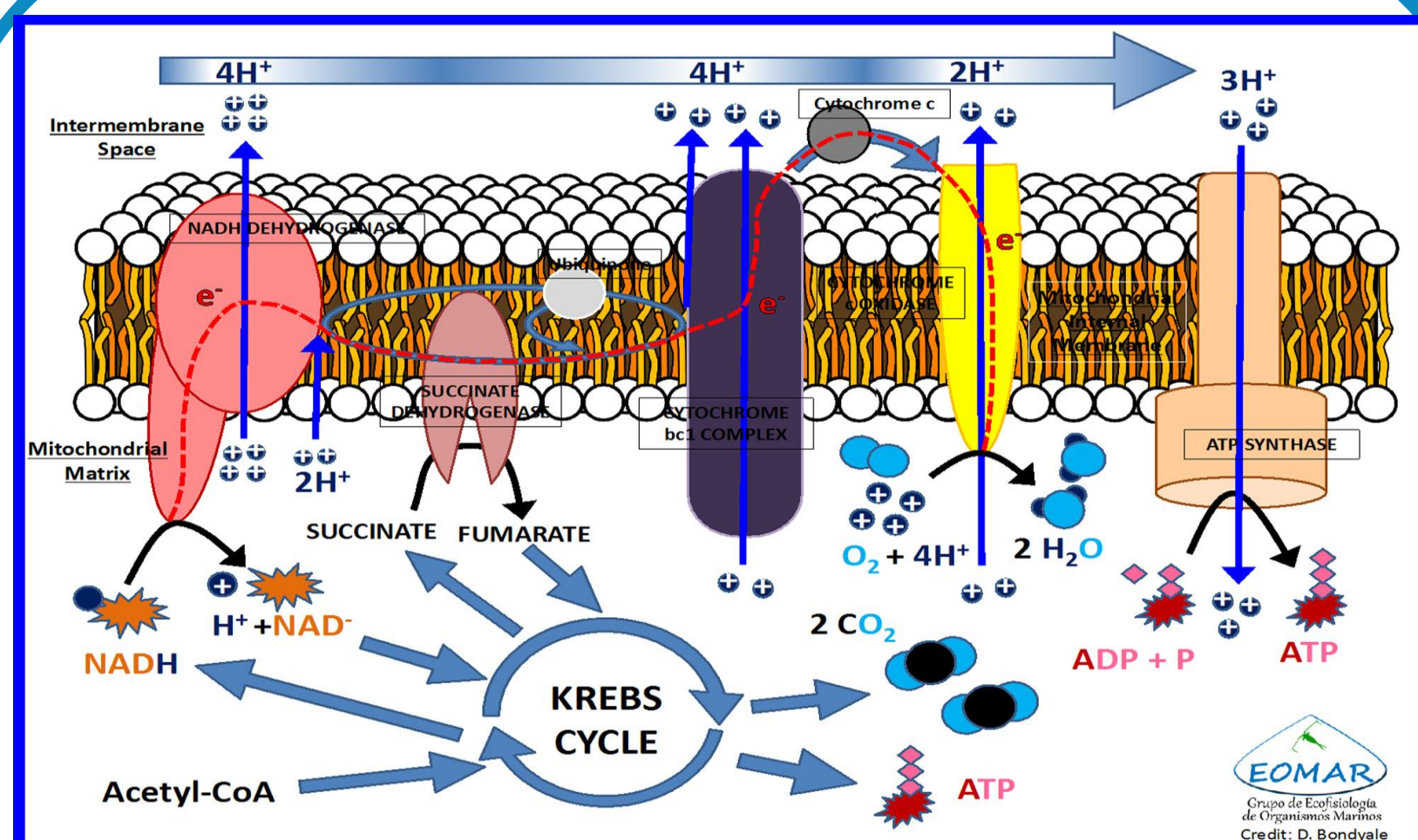
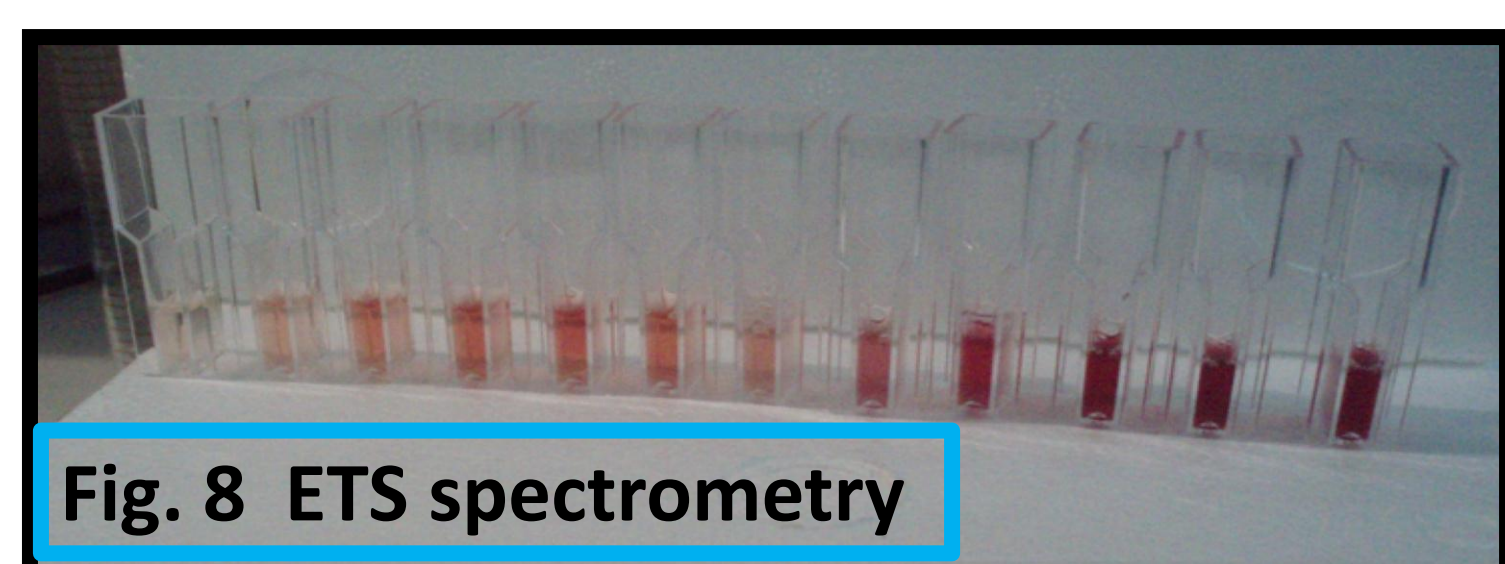
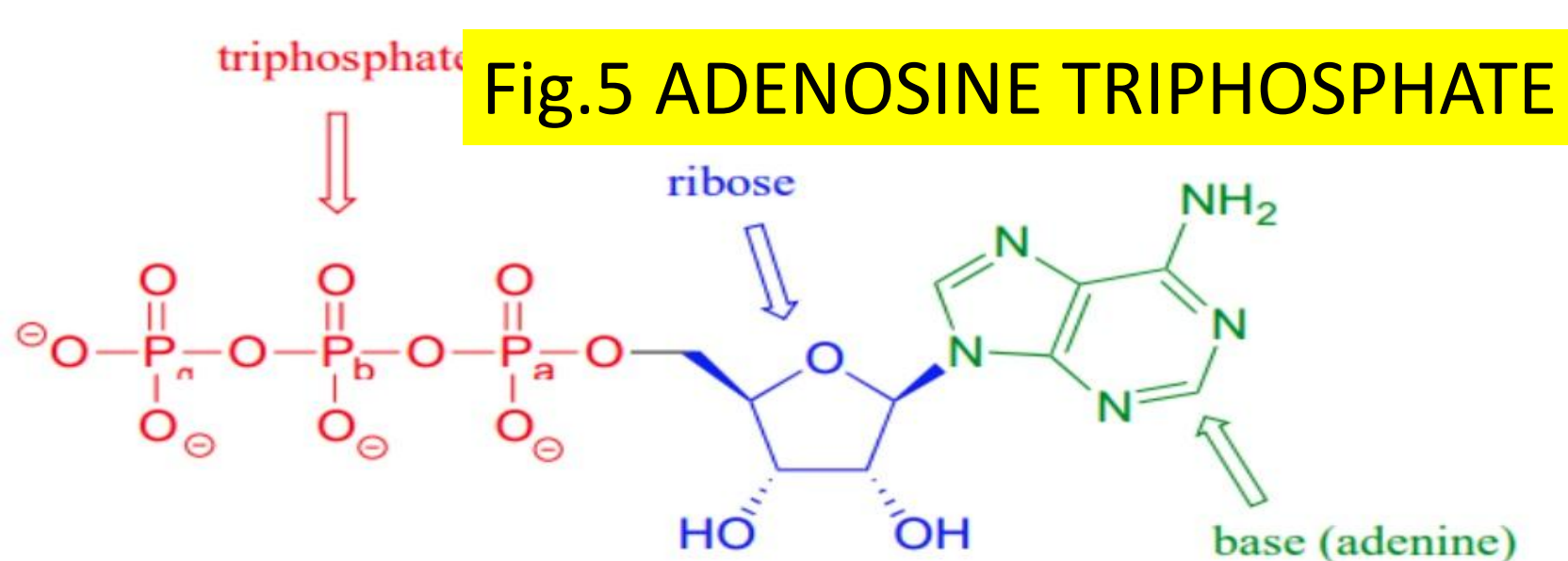
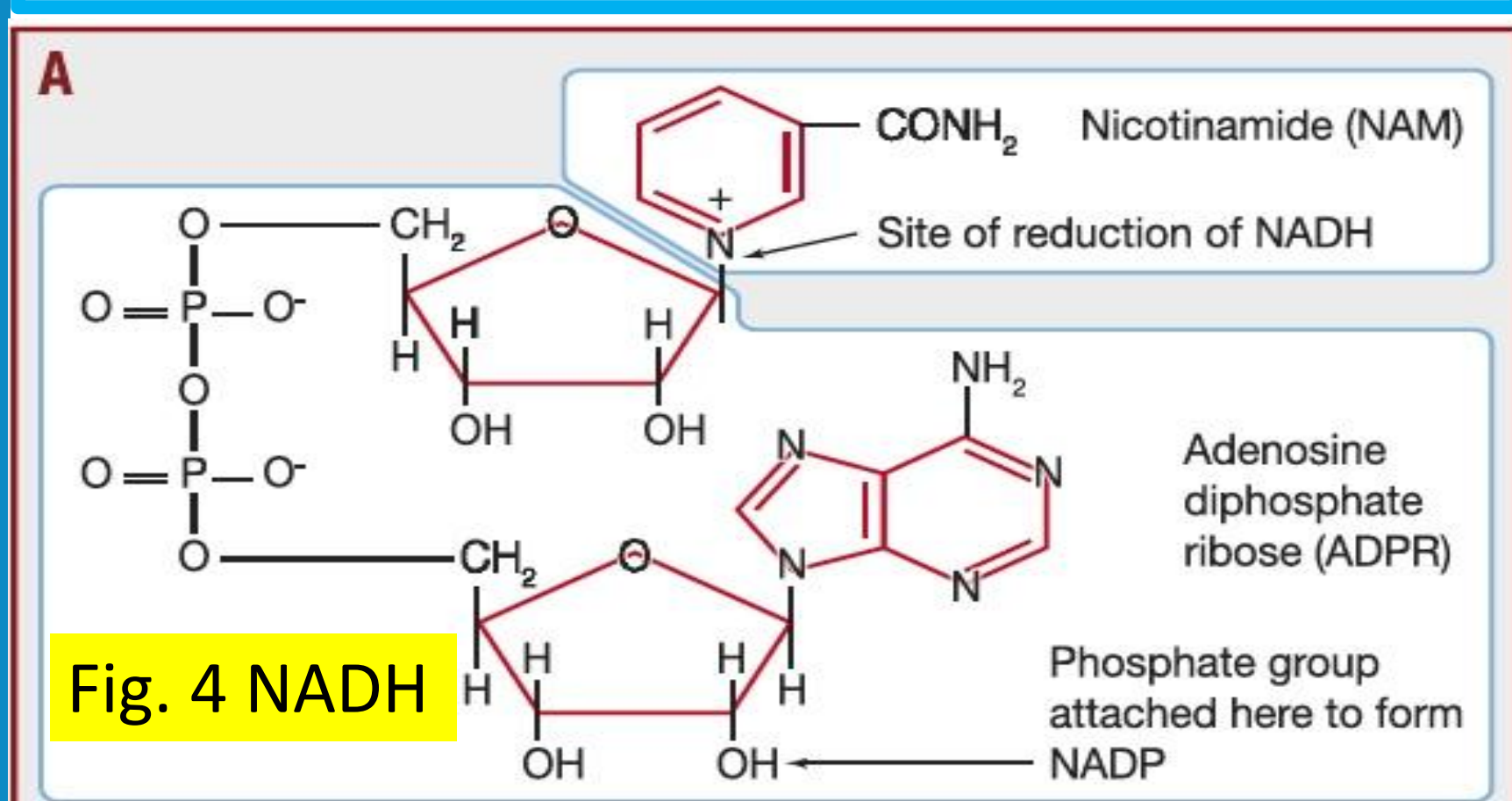


## BACKGROUND



**Fig. 1. ETS complexes (4) & ATP-producing enzymatic motor found in all living cells. This is the respiratory Electron Transport (transfer) System. Note proton pumping by 3 complexes and proton re-entry via the motor (ATPase).**



## ACKNOWLEDGMENTS:

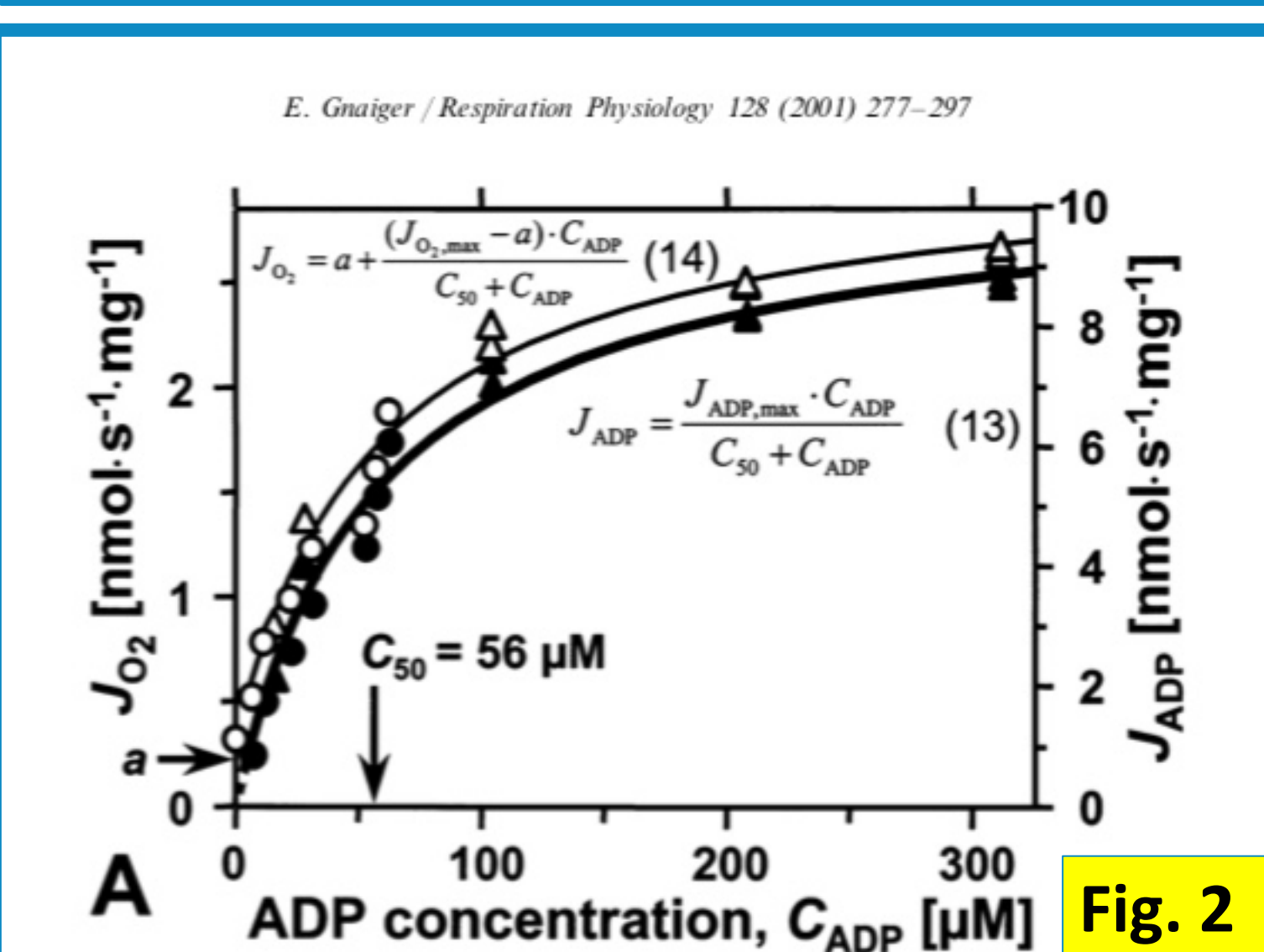


## PREMISE

1. Jellyfish metabolism needs incorporation (sensu oceanography) in marine ecosystems models.
2. Putting a few jellyfish in bottles and measuring their O<sub>2</sub> use (sensu marine biology) will not provide adequate data.
3. Biochemical sensing, kinetic analysis, and computer modeling must be employed to give requisite data acquisition rates.

### BOX 1 Metabolic states of mitochondria (Chance and Williams, 1956; Table V).

State	ADP [O <sub>2</sub> ] level	Substrate level	Respiration rate	Rate-limiting substance
1	>0 low	low	slow	ADP
2	>0 high	~0	slow	substrate
3	>0 high	high	fast	respiratory chain
4	>0 low	high	slow	ADP
5	0 high	high	0	oxygen



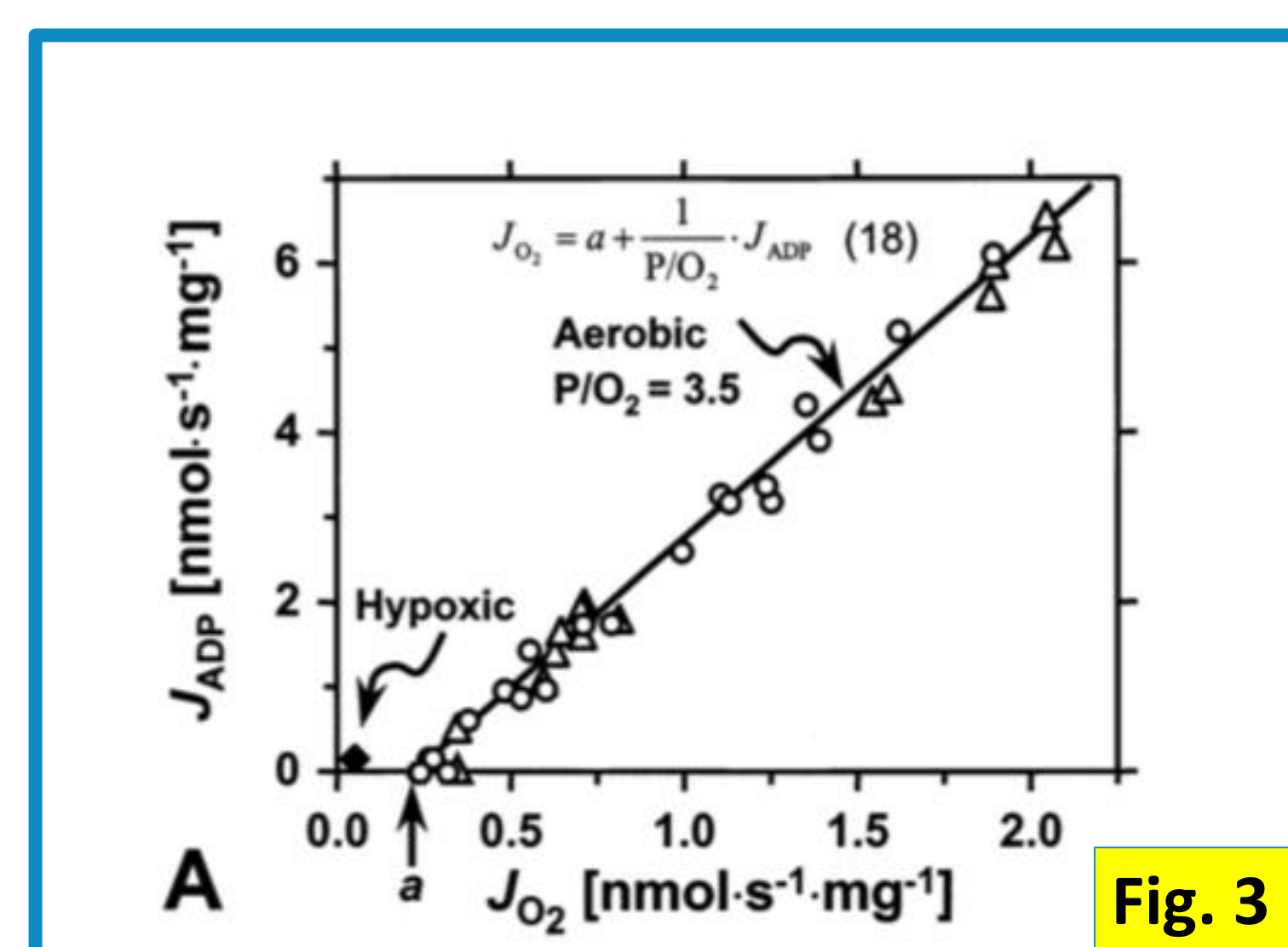
**Fig. 2 Caption: Michaelis-Menten dependence of both respiration (J<sub>O2</sub>) & ADP phosphorylation (J<sub>ADP</sub>) on ADP concentration. Note that the K<sub>m</sub> of J<sub>O2</sub> for ADP is 56 μM.**

## VISION

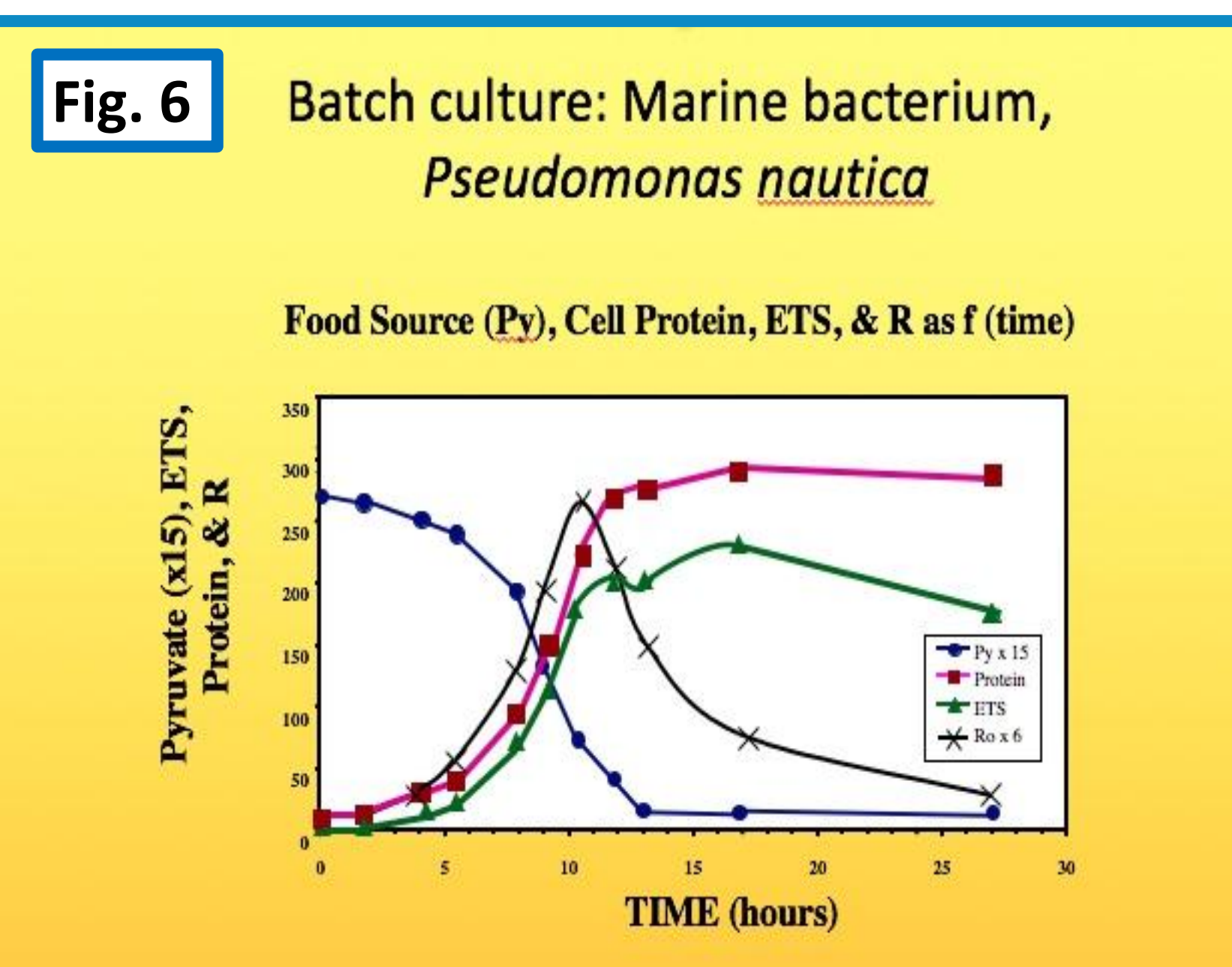
By extrapolating understanding from biochemical experiments on respiratory control in other organisms we can develop hypotheses about respiratory control in Jellyfish.

## VISION BACKGROUND

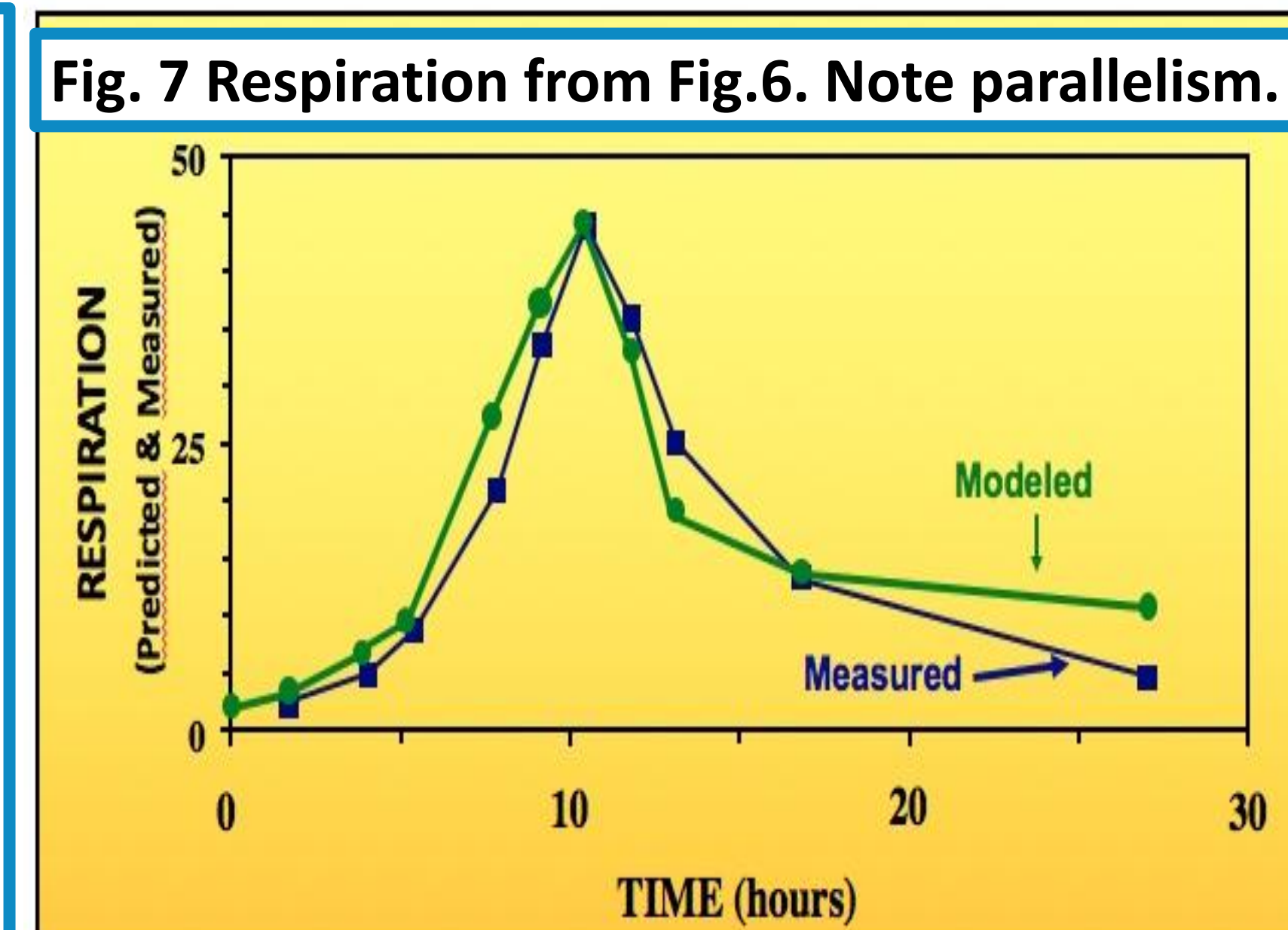
1. Accordingly, we know from the respiratory ETS (Fig. 1) experiments of Chance and Williams (Box 1), Jacobus et al., and Gnaiger (Figs. 2 & 3) that ADP levels in the vicinity of cytochrome oxidase stimulates respiration (J<sub>O2</sub>) when O<sub>2</sub>, substrate (NADH, Fig. 4) are present. In this situation, ADP (Fig.5), as the index of metabolic demand, drives J<sub>O2</sub> (Fig. 2 & 3). Analogy: "Demand-side" economics.
2. Pyridine nucleotide availability (mainly NADH) represents the "supply-side" of respiratory control and was explored through modelling (Packard et al., 1996). A theoretical enzyme-kinetic model of Pyridine nucleotide availability in bacteria cultures shifting from nutrient-sufficiency through nutrient-limitation to nutrient-starvation (Fig. 6) successfully predicted J<sub>O2</sub> in all physiological phases of the cultures (Fig. 7).



**Fig. 3. Caption: Linear relation between the ADP phosphorylation rate (J<sub>ADP</sub>) & the respiration rate (J<sub>O2</sub>). This permits calculation of the J<sub>ADP</sub> from respiration. Note that J<sub>ADP</sub> is equivalent to the Heterotrophic Energy Production (Packard et al., 2015).**



**Fig. 6 Caption: Time-course, pyruvate (Py) nourished culture exhausting Py and becoming starved. Note uncoupling between respiration (R) and potential respiration (ETS) as Py approaches zero. Note too, the parallel decline in R and Py after a 5 h lag.**



**Fig. 7 Caption: First-principles enzyme kinetic respiration model (EKM) based on ETS activity (Fig.8) & data from Fig. 6. Unlike Metabolic-Theory-of-Ecology modeled R, EKM-modeled R matched measured R in both nutrient-sufficient & nutrient-starved cells (Aguar et al., 2012)!**

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## FUTURE MODELLING

Modelling respiration with an EKM until nutrient limitation becomes extreme is the way to start. Then , the model must include an additional, ADP-dependent term that is activated at zero nutrients. It would mean that ETS activity (Fig. 8), ADP (FIG. 5), NADH (Fig. 4), and their K<sub>m</sub>s would need to be measured in seawater.

## Hypothesis

Respiration is controled by the ETS respiratory capacity when ETS substrate levels are not limiting, but when starvation commences, respiration is substrate controlled. Later when the externally derived substrates are exhausted, ADP controls the respiration rate.